

Aus dem Pathologischen Institut der Universität Basel

(Vorsteher: Prof. Dr. med. A. Werthemann)

Arbeit unter Leitung von Prof. Dr. med. S. Scheidegger

METATARSALGIA ANTERIOR

Morten's Disease

INAUGURAL-DISSERTATION

zur Erlangung
der Doktorwürde der Medizinischen Fakultät
der Universität Basel

vorgelegt von

JANETTE BENTLEY-JOHNSTON

von Sydney, Australia

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In January 1876 Thomas G. Morton first published his account of 'a painful affection of the foot, involving particularly the fourth metatarsal-phalangeal articulation', a condition which has since become known as Morton's Neuralgia etc., (which is not, however, to be confused with D. Morton's Triad).

Morton describes the pain as being neuralgic in character and sometimes so intense that the patient is obliged to take off his shoe and manipulate his toe. He believed the pain to be due to the compression of the nerve between the fourth and fifth metatarsal bones, this being facilitated by the greater physiologic mobility of these bones in comparison with the other metatarsal bones, and further by the shortness of the fifth metatarsal, which allows greater proximation to the fourth.

In Morton's series women showed greater predominance, although he later states in 'Surgery in the Pennsylvania Hospital' that the cases are more evenly divided between the sexes. He considers one of the main causes to be tightly fitting shoes. The condition was always found in an otherwise healthy appearing foot, where the patient tended to be overweight. For cases which did not respond to conservative treatment Morton recommended resection of the metatarsophalangeal joint and the tissues surrounding it including the nerves. He does not note any abnormality or structural change.

Since Morton's report in 1876 many articles have confirmed this condition as a clinical entity distinct from other types of metatarsalgia. In 1891 Bradford reported a certain amount of success with conservative treatment. He recommended stimulation of the circulation with hot and cold baths, local injections of cocaine and rest. In 1893 Hoadley exposed the digital branches of the plantar nerve to the fourth toe and found a neuroma which he resected. He then suggested a more conservative approach and in the case of surgery resection of the nerve rather than the joint as practiced by Morton. In 1898 Jones and Tubby reported 'neuritis of the digital nerves', but held to the older method of surgical treatment.

It was not until 1940, when Betts in Australia reported a series of 19 cases of Morton's toe, where a neuroma had been resected in each instance and histologically examined that the pathological basis of this condition was recognised. In all cases the fourth and not the fifth nerve as originally thought by Morton, was involved. Betts also noted that the condition was much more common in private practice, a fact that Tubby had also reported. Betts states that the pain was in the lateral third and the medial fourth toe, where sensory disturbances were often evident. He also noted occasional cascular and trophic diturbances.

Betts' theory as to the cause of neural damage differed from Morton's. He considered that the pain being relieved by the removal of the shoe and compression and flecion of the toes pointed to a stretching of the nerve rather than compression. This stretching he explained by the anatomical peculiarity of the fourth digital nerve, which is formed by the most lateral branch of the medial planter nerve and a communicating branch of the lateral plantar nerve. Each of these branches comes from the opposite side of the flexor brevis digitorum, which they cross to unite. After uniting to form the fourth nerve, it divides shortly distal to the transverse ligament to send a branch to the medial fourth and to the lateral third toe. It is therefore thicker than the other digital nerves owing to its double origin, and according to Betts has a thicker fibrous sheath. This thicker part lies immediately under (or on) the transverse ligament. While walking the nerve is anchored proximally by the contracted flecor brevis and dorsiflexion of the foot leads to it being stretched around the transverse ligament. Once neuritis develops there is little tendency for natural cure as the stretching across the the transverse ligament provides the necessary factor for a vicious cycle to develop. Chronic neuritis results in time with the formation of neuroma. Betts refers to a fibroneuroma. He compares this with the formation of late ulnar neuritis.

In 1943 Betts' findings were confirmed by McElvenny in America who, working independently found neuroma in cases with Morton's metatarsalgia. Histologically these tumors resembled neurofibromata, angiofibromata, and even glomus tumors. Like Betts he believed trauma to be most significant in the formation of

these tumors, although he says it must not be overlooked that neurofibromata arise spontaneously in many parts of the body and that the location of these tumors in the 3-4 cleft provides favourable conditions for pressure against the tumor. He also notes that these tumors occur in other digital clefts, but it is only when they are in the 3-4 cleft that a typical history of Morton's metatarsalgia is found.

Later a new aspect of Morton's metatarsalgia was brought forward by Nissen in England, who believed the nerve changes to be secondary to ischemia. In his series of 27 cases, women accounted for 21. In 8 patients the condition was found bilaterally. The youngest age of onset was 14 years. He states that the artery rather than the nerve is more liable to injury as the artery must pass through a fibrous tunnel in the transverse ligament in order to reach the plantar surface of the foot, where it is susceptible to pressure through the sole. An important variation of the arterial pattern is the origination of the artery from the 2-3 cleft, so that it must cross the common flexor sheath to reach the 3-4 cleft. In this instance the artery is particularly liable to impact from the sole.

Nissen also argues that there is little danger of the nerve being pinched or subjected to pressure by the bones. This he has been able to verify by taking radiographs with wire laid alongside the nerves. He also points out that the communicating branch of the nerve, which is necessary for the explanation of Betts' theory, is not always present. However, there is only one case in the literature where it has been reported that it was missing. In some cases too it is not possible for the surgeon to make an exact anatomical evaluation of the plantar structures, as they may prefer the dorsal or web-splitting approach. Nissen found the most constant symptom was pain on firm upward pressure in the sole of the foot. Anaesthesia was not always marked, probably due to take-over mechanisms by adjacent nerves. In most cases the foot appeared healthy, although some patients were noted with hallux valgus and anterior flat foot. Where treatment was not successful or only partly so re-exploration often showed similar changes in other clefts. Similar nodules in the course of the median and ulnar nerves are also found in Volkmann's contracture, where the nerve lesions are undoubtedly caused by ischemia.

An interesting aspect is brought forward by Watson Jones. In a case of pleonosteosis which he attributes to capsule contracture rather than changes in the epiphyses he reports bilateral median palsy and bilateral Morton's Metatarsalgia with large digital neuroma. In acromegaly cases of median palsy have been reported, and according to Nissen there may also be plantar digital neuritis. This he thinks may be due to the thickening of the transverse ligament with compression of the digital artery in the same manner as the median nerve is compressed by the carpal ligaments.

Mulder in Holland advances yet another theory on the mechanical causation of this condition. He believes a weak transverse arch to be the basic metatarsal heads, which in turn leads to an enlargement of the intermetatarsophalangeal bursae. Thus, displacement of plantar tissue, including the nerve into the space occupied by the bursa is possible, and a pinching of the nerve between the metatarsal heads when the foot is compressed is explained. The artery may also be liable to the same trauma. Mulder, like Betts, observes the firm attachment of the nerve to a fibrously-thickened bursal sack.

Mulder's theory, it would appear, is a modified version of Morton's original concept of the nerve becoming compressed by mobile metatarsal bones, in this case, the increased mobility is not physiological, but pathological. Other writers, Betts, Morton etc., do not note this weakness, however, but comment on the healthy appearance of the foot, nothing that in general the patients tended to be overweight.

Nora in one article and Litchmann in another both speak of a neuroma which causes Morton's metatarsalgia. Earlier writers have not been of this opinion, although they have noted neuromas as being constant findings in all cases. With the increasing literature on the subject, there seems to have arisen some confusion as to cause and result. Pain and the formation of neuroms are the result of neural damage, the former being the immediate result, the latter the long-term result. Betts speaks of chronic irritation causing neuritis, which explains the neuralgic pain and its tendency to be brought on even by quite minor trauma. He describes neuroma as a later development. It is possible, however, that once the neuroma develops, that the nerve by its very struc-

ture becomes increasingly susceptible to trauma but the neuroma as such is not the cause of Morton's metatarsalgia.

Pathology of the Lesion

Since a neuroma was first found in cases of Morton's metatarsalgia there has been little agreement among pathologists as to whether it is a true neoplasm or a degenerative condition of the nerve. In the case of degeneration it is not clear whether arterial changes are primary or secondary to those of the nerve.

Betts reports neuritis resulting in fibroneuroma. Macroscopically the largest neuromas were the size of a pea with a well defined bursal sack around them. McElvenny, in his five cases, describes changes which resemble neurofibromata or false neuroma, angiofibroma and glomus tumor. He however, says that the tumors had not yet definitely been classified. He notes central cystic formation in a few of the lesions.

King does not consider neuroma to be true neoplasms and calls them 'sclerosing neuroma' to differentiate them from amputation and traumatic neuroma. He describes reduction in the number of nerve fibers and an increase in connective tissue, absence of lymphocytes and polymorphonuclear leucocytes, increase in the number of blood vessels and hyaline thickening of their walls. He considers these changes to be a type of reactive hyperplasia with sclerosing and degenerative changes.

Bickel refers to 'tumorfactive fibrosis' to express both degenerative and proliferative changes. He also differentiates early and late changes of the nerve trunks and the surrounding tissue sheaths. The commonest early nerve change is interstitial oedema, associated with demyelisation and swelling of the entire nerve trunk. Shortly afterwards this is accompanied by proliferation of the neurilemma nuclei, giving the picture of pronounced cellularity. This is in some cases accompanied by cyst formation. Perineural oedema with proliferation of connective tissue fibroblasts results in collagen tissue which is later replaced by hyaline. The end result is a perineural scar.

Bickel further states that all the lesions appear vascular, the vessel walls being oedematous, suggesting trauma. Later the vessels were noted as being sclerotic with endarteriitis and occasionally thrombosis. Here it is difficult to say whether the arterial or neural changes are primary, but it does appear that nerve changes have taken place while relatively good vascularity was present. However, Nissen describes arterial degeneration in early cases of Morton's metatarsalgia where the nerve lesion is not pronounced and he does not note any vascularity. The nerve capsule or bursal sack he considers to be of periarterial origin. McElvenny's description of angiofibroma suggests vascularity which like King's and Bickel's descriptions would seem to contradict Nissen's theory that ischemia was primary to neural change.

In control studies Ringertz found similar vascular patterns in normal feet as those reported by Nissen. Nissen's theory also does not explain why vascular degeneration occurs mainly in woman, often of the younger age groups, who are otherwise are not prone to arterial degeneration.

Nora was able to show in a control group that neuromas are not an uncommon lesion in the interdigital space. In a series of autopsies where the patients had no history of foot pain, he was able to demonstrate lesions which were macroscopically and microscopically identical to those in Morton's metatarsalgia. He does not state, however, whether all interdigital clefts were investigated or specifically the 3-4 cleft. It is the special location of the neuroma, as McElvenny pointed out, which makes the condition particularly painful. Surgeons have previously reported neuromas in other clefts, where pain was less severe.

In our case a 35 year old female nurse had a typical history of shooting pain from the 3-4 cleft which radiated along the nerve and which was intensified by dorsiflexion of the toes. The condition was bilateral. After resection of both fourth digital nerves, the patient had no recurrence of symptoms.

Macroscopically the nerves showed moderate thickening and were not nodular. Microscopically degenerative and proliferative processes were to be seen in both the nerves and vessels. There was pronounced oedema of the nervous tissue and in some places demyelisation and proliferation of fibrils were present. The nerve

sheaths were enlarged and showed slight inflammatory reaction (King does not note any such reaction and this is one of the reasons why he separates this lesion from traumatic neuroma). In some places intima proliferation has led to complete occlusion of the arterioles. There was no inflammatory reaction of the blood vessels. The changes in the walls of the blood vessels appear to be of an earlier date than those of the nerve. There were no changes to suggest a neoplasm and in this respect the generally used term 'neuroma' is misleading. A better description would be hypertrophic neuritis.

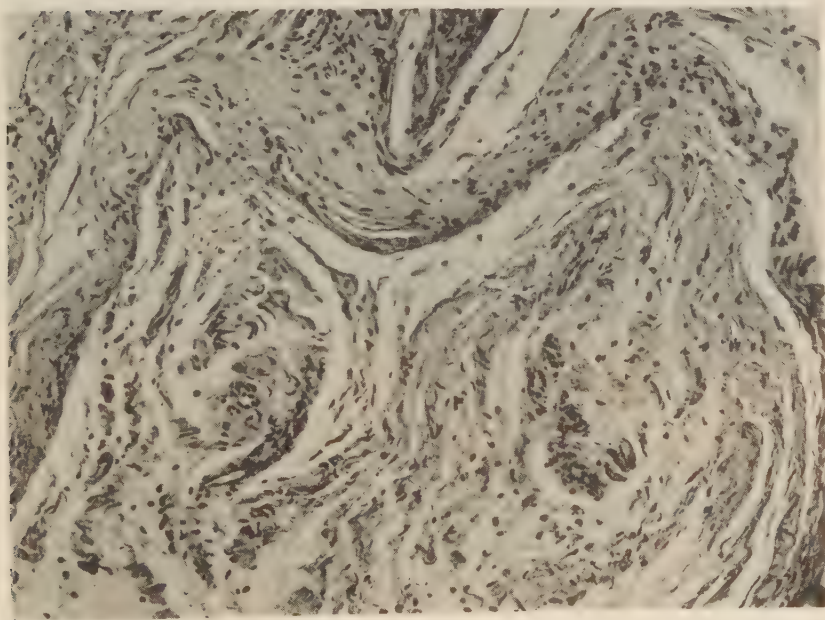


Fig. 1

Degeneration of the nerve fibers with thickening of the nerve sheath, oedema and disintegration at the centre.

1 : 120

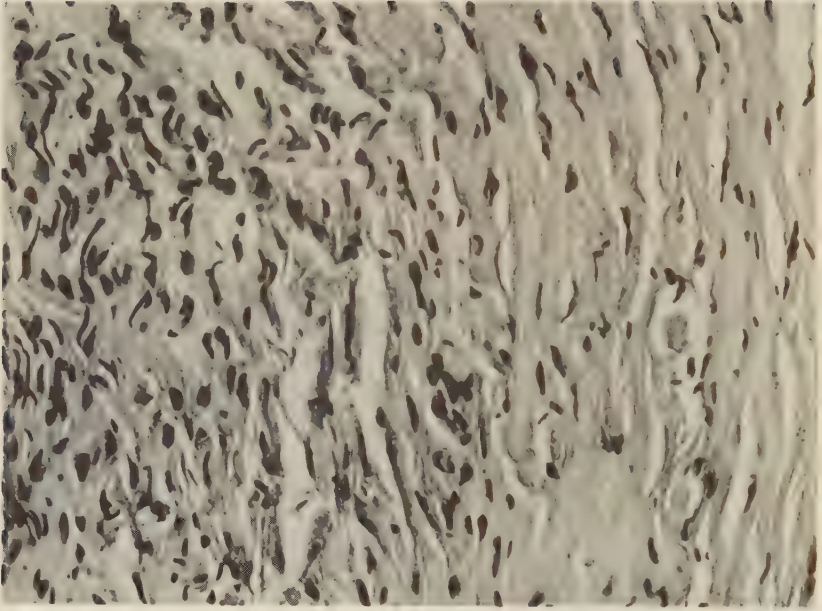


Fig. 2

Pronounced thickening of the nerve sheath with hyaline degeneration. 1 : 300

Our findings suggest that a disturbance in circulation is primary to neuritis which would support Nissen's theory. However, why similar arterial changes occur without 'neuroma' is unclear.

Discussion

Why the condition occurs on one hand so commonly among nurses, who wear a sensible type of shoe, and on the other hand in people wearing fashionable narrowing fitting shoes is not quite explained. In the case of the nurses a pinching of the digital nerve seems rather unlikely. In the case of the fashionable shoes so often reported perhaps the high heel rather than the narrow fit is responsible for the trouble. Weight bearing, which is constant in the case of nurses and falsely distributed in the case of wearers of high heeled shoes, probably leads to arterial damage. Nissen believes the artery to be more liable than the nerve to pressure through the sole.

No one single factor seems common to all cases at all times. No doubt different factors are first necessary to bring about neuritis (for instance ischemia) and secondarily to produce an attack (for instance stretching of the nerve as thought by Betts, or pinching of the nerve as originally thought by Morton).

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